epithelial cells was significantly higher in the samples from the children deficient in vitamin A (table). Vitamin A intake was less than the recommended allowance of 400 retinol equivalents/day in all three groups; the average intake was lower in groups 2 and 3 than group 1. Groups 2 and 3 had a lower weight for height than group 1, but the differences were not significant.

Comment

The increased bacterial adherence to epithelial cells found in children with vitamin A deficiency provides evidence for an additional factor that can contribute to the increased risk of infection in this disease. Nutritional deficiencies impair several host defence mechanisms, but much of the work done in the past 20 years focused on immune responses rather than physical barriers.[1 3]

This study shows that respiratory epithelial surfaces of vitamin A deficient subjects may permit increased colonisation and thus allow penetration of mucosa, leading to systemic infection. Various barriers contribute to local defence on mucosal surfaces, including mucus, glycocalix, cilia, secretory IgA, neutrophils, macrophages, and T lymphocytes. It is not clear which aspects of this barrier are affected by vitamin A deficiency. A prospective study should be done to correlate bacterial adherence to epithelial cells with morbidity. A firm cause and effect relation would be established if vitamin A supplements were found to correct the abnormal bacterial adherence.

1 Chandra RK. Nutrition, immunity, and infection. Present knowledge and future directions. *Lancet* 1983;i:688-91.
2 Sommer A, Tarwotjo I, Hussaini G, Susanto D. Increased mortality in children with mild vitamin A deficiency. *Lancet* 1983;ii:585-8.
3 Vyas D, Chandra RK. Vitamin A and immunocompetence. In: Watson RR, ed. *Nutrition, disease resistance and immune function*. New York: Marcel Dekker, 1984:325-44.
4 Wong YC, Buck RC. An electron microscopic study of the metaplasia of the rat tracheal epithelium in vitamin A deficiency. *Lab Invest* 1971;24:55-66.
5 Niederman MS, Rafferty TD, Sasaki CT, Merrill WW, Matthay RA, Reynolds HY. Comparison of bacterial adherence to ciliated and squamous epithelial cells obtained from the human respiratory tract. *Am Rev Respir Dis* 1983;127:85-90.

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Risk to Metropolitan police officers from exposure to hepatitis B

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Since 1981 St Thomas's Hospital has provided a service to members of the Metropolitan police who have been in incidents conferring a risk of exposure to hepatitis B virus. We describe our experience in assessing this risk and provide recommendations for immunising police officers.

Subjects, methods, and results

In the procedure followed by the police after an incident conferring a risk of hepatitis police surgeons take a blood sample from the officer and, whenever feasible, from the person suspected of having hepatitis (the contact). The officer then attends the virology department, where a doctor assesses his risk and obtains details of the incident and the risk factors.

Samples from officers and, when available, the contact were tested for hepatitis B surface antigen (HBsAg); those found positive were tested for hepatitis B e antigen (HBeAg) and antibody. All samples from officers that were negative for HBsAg were tested for antibody to HBsAg. (If there had been a considerable delay between the incident and presentation specimens from contacts were initially tested with a modified Hepatest II (Wellcome), which gave results within one hour.) If the suspect was positive for HBsAg the officer was offered hepatitis B immunoglobulin if his eyes or fresh cuts had been contaminated by blood or saliva or after a needlestick incident. When hepatitis B vaccine became widely available it was recommended in addition. A second dose of immunoglobulin was given one month later unless the contact had antibody to HBeAg or hepatitis B vaccination had been started within one week of the incident. When blood from the contact was not available offering immunoglobulin depended on the risk and on the availability of immunoglobulin.

During 1981-6, 503 incidents with a risk of hepatitis were reported in 793 officers, of whom 248 (31%) were given hepatitis B immunoglobulin. The number of officers seen increased considerably, from 46 in 1981 to 187 in 1986. During 1983-6, 33 of 198 samples tested from suspects were positive for HBsAg and half of those were also positive for HBeAg. Most contacts (table) were drug abusers or dealers, whereas the most frequent type of incident was contact with a bleeding person. Altogether 12% of officers attended because they had arrested a person who then claimed to be a carrier. Other contacts included prostitutes, vagrants, transvestites, and corpses. No officers were found to be HBsAg positive and only three (1%) of 425 tested had evidence of past infection. No subsequent attacks of hepatitis B were identified in any of these officers.

Comment

Hepatitis B can be transmitted easily through parenteral contact with infected blood and secretions, and such cases occur in police officers.[1] The number

Details of contacts involved in incidents conferring risk of exposure to hepatitis B to Metropolitan police officers, 1983-6

	Contact					Incident				
	Male homosexual	Drug addict or dealer	Homosexual drug addict	Ethnic origin suggested high risk of carriage of hepatitis B virus	Not known or other	Minimal contact but officer concerned	Bleeding person	Aggressive contact (contact not bleeding)	Needlestick injury	Other
1983	1	72	3		20	13	24	16	38	5
1984		54		2	12	10	19	19	17	3
1985	4	73	2	1	16	11	34	20	27	4
1986	10	100	3		22	13	50	41	23	8
Total (%)	15 (4)	299 (76)	8 (2)	3 (1)	70 (18)	47 (12)	127 (32)	96 (24)	105 (27)	20 (5)

of officers at risk increased during our study; this probably reflects increases in urban violence and the number of drug abusers. Nevertheless, few officers had markers of hepatitis, which suggests that unreported exposure is uncommon.

A central service for the Metropolitan Police Force seems to work well; police officers and surgeons generally know the correct course of action after exposure, and prophylaxis can be given when it is most likely to be effective. An increasing problem is unavailability of blood from contacts, particularly drug abusers, who are often uncooperative and may have sclerosed veins; this may result in costly hepatitis B immunoglobulin being given unnecessarily. Vaccination of police officers would be effective in reducing the risk of acquiring hepatitis B but would be expensive; at £32.60 per course it would cost over £880 000 to vaccinate the 27 000 officers in the Metropolitan police alone. The group most at risk is officers on ordinary street duty, some of whom have already been vaccinated by their general practitioners. Most officers can avoid contamination with hepatitis B by adopting precautions such as taking care with needles and wearing gloves when dealing with bleeding accident victims. For the others a service such as ours seems the most cost effective in preventing hepatitis B.

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1 Peterkin M, Crawford RJ. Hepatitis B vaccine for police forces? *Lancet* 1986;ii:1458.

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Menstrual and ovulatory disturbance in bulimia

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Anorexia nervosa is associated with menstrual disturbance.[1] This is caused by hypothalamic dysfunction except when weight falls to below 70% of the mean weight of a matched population, when disturbance of pituitary function occurs.[2,3] The cause of this hypothalamic-pituitary disturbance is not clear but a fall below a critical level of body fat is the most popular theory.

Women with bulimia at normal body weight have also been reported to suffer menstrual disturbance, but the cause is not known.

This preliminary study aimed at confirming this disturbance in women with bulimia, assessing the effect on ovulation, and investigating the underlying hormonal changes.

Patients, methods and results

Fourteen women aged 18 to 40 years who were within 10% of the mean weight of a matched population (taken from weight charts for a normal population) and were not taking the contraceptive pill entered the bulimic treatment programme during the study. The average age was 25 (range 16-46), mean weight 102% of the mean matched population weight (range 90-110%). Binge eating and vomiting were reported on average five times a week (range 1-21 and 0-21 respectively) and mean duration of illness was five years (range 1-11). Of the sample, five women (36%) had regular menses (cycles over the past six months not varying in duration by more than 10 days from one to the next), six (43%) irregular (at least one period in six months but cycles varying by more than 10 days), and three (21%) absent (no periods in six months). The sample had a history of fairly stable recent weight, no patient having gained or lost more than 10% in weight during this time.

Three serum progesterone assays taken at two week intervals showed that two patients had ovulated and 10 had not, and two patients yielded equivocal results. Daily temperature charts showed that one of these last two (case 4) had ovulated during the study, while the other had not (case 10).

Of the 11 (79%) patients who had not ovulated, three refused further investigation. The rest underwent a luteinising hormone releasing hormone stimulation test, entailing serial estimations of serum luteinising hormone and follicular stimulating hormone concentrations after intravenous injection of 100 μg of luteinising hormone releasing hormone analogue. The results were interpreted according to the guidelines established by Bergh *et al* and Franks *et al*.[3,4] All eight patients also underwent an ultrasound scan of their ovaries (appearance classified according to the criteria of Adams[4]) and a progesterone challenge (menstrual type bleeding on withdrawal after five days of medroxy progesterone acetate 5 mg daily indicates an endometrium primed by oestrogen).

Clinical details of 14 patients studied

Case No	Menstrual pattern	Ovulation	Ovarian morphology	Site of dysfunction
1	Amenorrhoea	No	Normal	Hypothalamus
2	Irregular	No		Refused further investigation
3	Irregular	No	Not investigated	Pituitary
4	Regular	Yes		
5	Regular	Yes		
6	Amenorrhoea	No	Polycystic	Ovaries (polycystic)
7	Irregular	No	Not investigated	Hypothalamus
8	Regular	Yes		
9	Irregular	No	Multicystic	Pituitary
10	Irregular	No	Normal	Hypothalamus
11	Regular	No	Normal	Pituitary
12	Amenorrhoea	No	Normal	Hypothalamus
13	Regular	No		Refused further investigation
14	Irregular	No		Refused further investigation

One patient (case 6) yielded a test result consistent with the presence of polycystic ovaries, which was confirmed by ultrasound. Three patients (cases 1, 7, 12) showed a pattern consistent with hypothalamic dysfunction (normal low baseline gonadotrophins with a pronounced response of luteinising hormone to stimulation). One (case 10) showed an intermediate response that we took to indicate hypothalamic dysfunction. Three patients (cases 3, 9, 11) showed a pattern consistent with pituitary dysfunction (poor response to luteinising hormone releasing hormone stimulation), and all three showed positive responses to progesterone challenge, indicating that lack of oestrogen priming was not the cause of the result. Prolactin concentrations were normal in all patients (table).

Comment

Nine (64%) of our sample suffered menstrual irregularity or amenorrhoea and 11 (79%) failed to ovulate